

# Adult Stature and Risk of Cancer<sup>1</sup>

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## ABSTRACT

We examined the relationship between adult stature and cancer incidence using data from the first U. S. National Health and Nutrition Examination Survey and its follow-up study. Among 12,554 participants 25-74 years old, 460 cancers occurred in men and 399 in women after an average follow-up period of approximately 10 years. The age-adjusted relative risk of cancer for the second (Q2) through fourth (Q4) quartiles of stature compared to the first quartile among men were significantly increased: 1.5, 1.4, and 1.4. After adjustment for race, cigarette smoking, income, and body mass index, the all-sites cancer relative risk increased slightly to 1.6, 1.5, and 1.6. For most cancer sites in men, and particularly colorectal cancer (relative risk = 2.1 for Q4), the lowest incidence was observed among those in the shortest quartile of stature. A weaker, positive association was evident among women, restricted primarily to cancer of the breast and colorectum (relative risk in Q4 = 2.1 and 1.6 for the two cancers, respectively). These findings indicate that short stature is associated with reduced risk of cancer, particularly in men, and suggest a role for nutrition early in life in human carcinogenesis.

## INTRODUCTION

Investigations of the relationship between body size and cancer have focused primarily on weight and body mass indices. Several studies demonstrate increased rates (or risk) of cancer among heavy or overweight individuals (1-4), while others show lean persons to be at increased risk (5, 6). Recent reviews of a large number of such investigations have attempted to clarify the findings on the basis of factors associated with leanness (e.g., cigarette smoking or undiagnosed illness) in support of a positive body weight-disease relationship (7, 8). This would be consistent with numerous animal experiments demonstrating that mortality and tumor incidence are directly related to adult body weight and daily caloric intake (9, 10). While intake in excess of energy requirements and adiposity or obesity are strongly implicated as causes of cancer by these studies, little evidence exists concerning the relative contribution of other specific body components, such as lean tissue mass, stature or body length, or frame size, to the observed relationships.

In this regard, stature (*i.e.*, standing height) has been less actively investigated in epidemiological research than body weight or fatness. Being related to nutritional exposures (*e.g.*, macronutrient intake) occurring up to the time of maturation and remaining relatively fixed thereafter, stature represents an important marker of the effects of early nutrition on human carcinogenesis. Previous studies have shown that breast and lung cancer cases were taller than noncases (2, 3, 11-16), and Hodgkin's disease (17), acute leukemia (18), and osteogenic sarcoma (19) have also been associated with increased stature.

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We analyzed data from NHANES I<sup>3</sup> and its epidemiological follow-up in order to determine whether stature is related to cancer incidence in men and women.

## MATERIALS AND METHODS

Between 1971 and 1975, 14,407 men and women aged 25-74 years participated in NHANES I, conducted by the National Center for Health Statistics and designed to characterize a variety of health and nutrition related parameters in a representative sample of the United States population (20). Of the participants, 12,554 were successfully traced and reinterviewed (14% by proxy) as part of the NHANES I Epidemiologic Follow-up Study during the period 1982-1984. Therefore, the analytic cohort included 5141 men (41%) and 7413 women (59%), of whom 84% were white. The mean age at study entry was 48 years for women and 52 years for men, and the average length of follow-up was 10 years.

Stature and sitting height were measured to the nearest millimeter and weight to within 10-g increments at the baseline examination using standard state-of-the-art methods (21). Leg length was calculated by subtracting sitting height from stature. The baseline evaluation provided information concerning family income and dietary intake (*i.e.*, a 24-h recall questionnaire), while cigarette smoking and gynecological histories were derived from both baseline and follow-up interviews.

Incident cases of cancer (excluding nonmelanoma skin cancer), confirmed through hospital record or death certificate review, served as study end points. Thirty-five cancer cases prevalent at baseline were excluded. Pathology reports were obtained for all cases. During the follow-up period, 460 cases occurred among men (114 lung, 95 prostate, 62 colorectum, and 27 bladder), and 399 among women (122 breast, 67 colorectum, 30 corpus uteri, and 20 cervix). Cancer of the colon and rectum were left combined because of small case numbers and similarity of their results. For all other individual cancer sites there were fewer than 20 cases in either sex, and these were combined within sex (170 in men, 168 in women). A few individuals with an incident cancer at more than one site were included as cases for each site separately, but only once in the all-sites (and other sites, if appropriate) analyses.

Sex-specific quartiles of stature, leg length, and sitting height as well as octiles of stature were determined based on the entire analytic cohort. Age-adjusted least-squares means of several baseline characteristics were calculated using standard linear regression model statistical software (22). Relative risk of cancer and 95% confidence limits according to stature categories were estimated using the proportional hazards method of Cox (22, 23). In all regression models adjustment was made for age, and where indicated in the text and tables, for other known risk factors (*e.g.*, cigarette use and body mass index). Test for trend in relative risk across height categories was based on the statistical significance of a trend variable in the proportional hazards model. Age-standardized incidence rates were also calculated using the direct method, with the entire cohort serving as the standard population.

## RESULTS

Stature was normally distributed in both sexes, with means (and standard deviations) of 173.8 (7.2) and 161.2 (6.5) cm for men and women, respectively. Mean stature decreased steadily with age, from 176.7 and 162.9 cm among 25- to 34-year-old men and women, respectively, to 171.1 and 158.3 cm in the 65- to 74-year age group. In contrast, annual cancer incidence increased exponentially with age, from 5.7 and 9.7/100,000

<sup>3</sup> The abbreviations used are: NHANES I, the first United States National Health and Nutrition Examination Survey; BMI, body mass index.

person-years (men and women, respectively) in 25- to 34-year olds, to 231.6 and 136.8/100,000 in 65- to 74-year olds.

The relationship between stature and several potential cancer risk factors is shown in Table 1. Among men, the proportion of white persons and current cigarette smokers increased somewhat with stature, as did body weight and total caloric intake. Age, the proportion of individuals with low annual family income, and BMI were inversely related to stature. Similar trends were evident for women with the exception of race, which showed no association.

Age-adjusted relative risks of cancer according to quartiles of stature are demonstrated in Table 2. The lowest overall risk was experienced by persons in the lowest stature quartile (*i.e.*,  $\leq 169$  cm in men or 157 cm in women), with a greater stature-cancer association being demonstrated among men. Age-standardized incidence rates (all-sites) for low to high quartiles were 808, 1206, 1098, and 1104 cases/100,000 men, and 540, 570, 554, and 595/100,000 women. The relationship between all-sites cancer and stature was similar across subgroups of age, race, income, smoking status, and BMI, and a generally comparable relationship was also observed for cancer mortality (not shown).

In an effort to exclude confounding by other potential cancer risk factors as a reason for these observations, we further adjusted for race, smoking status, family income, BMI, and in women, several gynecological factors. This increased somewhat the relative risk of total cancer associated with the second through fourth quartiles: to 1.6, 1.5, 1.6 in men and 1.2, 1.1, 1.1 in women. Further adjustment for dietary intake of total calories or fat did not alter these findings. Although most of the site-specific 95% confidence intervals included 1.0, relative risk estimates are generally greater than one in the three taller categories, with the exception of bladder cancer in men and cervical cancer in women, two sites having the smallest case numbers and the widest confidence intervals. The tallest men and women experienced a two-fold increased risk of colorectal and breast cancer, respectively. A positive linear trend was evident for breast cancer in women (test for trend,  $P = 0.01$ ).

Since the above observations suggest a threshold effect, particularly for men, a more detailed evaluation of the stature-cancer relationship was conducted. Cancer incidence according to sex-specific octiles of stature is plotted in Fig. 1. Incidence increased dramatically in men taller than 169 cm, confirming the threshold observed for the first quartile, and remained approximately 30–50% higher than the incidence among shorter men. Although no threshold is evident for women, there is a suggestion of increasing incidence in the taller categories.

Finally, we explored the two components of stature, *i.e.*, leg

length and sitting height, for possible differences in effect. The findings, shown in Tables 3 and 4, support an overall greater role for leg length, particularly in men, where a threshold pattern similar to that for stature is suggested. The data also demonstrate some risk elevation among women in the highest quartile of leg length for breast, colorectal, and cervical cancer.

## DISCUSSION

To our knowledge, this is the first report of a positive association between adult stature and total cancer incidence. In this investigation, individuals in the lowest quartile of stature developed cancer at a substantially lower rate than did taller persons, an association not accounted for by several known cancer risk factors. The effect was of greater magnitude among men and was evident in and relatively stable across nearly every subgroup of age, race, income, smoking status, and body mass index. Although a threshold effect predominated for most sites, a dose-response relationship was suggested for cancer of the breast in women.

We do not believe these findings are likely due to methodological biases or limitations. This was a prospective study of a representative sample of the United States population. The observed distributions of stature (a relatively fixed biological parameter in adulthood) are comparable to those from other large studies in developed countries (24). Stature was measured using standard methods under controlled conditions. Hospital records and death certificate review yielded thorough case ascertainment. Although site-specific case numbers are not large, decreased cancer incidence among the shortest individuals was evident for most sites. The latter finding also minimizes the possibility that residual confounding by other cancer risk factors (*e.g.*, cigarette use, or reproductive history) is responsible for the observed association. Finally, because we saw evidence for a secular trend in stature, age-specific quartiles were also used in separate analyses which gave similar results.

Findings of previous studies from several diverse populations (including Brazil, Canada, Finland, Great Britain, Greece, the Netherlands, Japan, and the United States) support the hypothesis that stature is positively associated with the risk of cancer of certain sites (2, 3, 11–19); however, there have been conflicting reports (25–30). Increased stature of breast cancer cases relative to noncases was limited to premenopausal women (31) or to postmenopausal Japanese women (but not Caucasians) (32) and was contradicted in other studies by an inverse relationship (25, 26). Two studies have reported no significant association between stature and prostate cancer (27, 28), although in one study the distribution of stature among cases was

Table 1 Age-adjusted mean<sup>a</sup> baseline characteristics of cohort according to quartile of stature by sex, United States, 1971–1984

Stature quartile	Stature (cm)	Wt (kg)	Age (yr)	% white	% with income <\$7000	% current smokers	BMI (wt/height (m <sup>2</sup> ))	Energy intake (kcal) <sup>b</sup>
<b>Males</b>								
1 ( $\leq 169$ cm)	164.8	70.0	58.2	83.4	39.9	38.6	25.8	1879
2 ( $\sim 173.8$ cm)	171.6	76.3	53.7	84.6	36.5	41.4	25.9	2086
3 ( $\sim 178.6$ cm)	176.1	79.4	50.9	85.8	31.8	44.2	25.6	2253
4 ( $> 178.6$ cm)	182.9	85.4	46.2	88.8	29.3	40.7	25.5	2441
<b>Females</b>								
1 ( $\leq 157$ cm)	153.1	62.5	54.2	83.8	43.3	28.6	26.7	1362
2 ( $\sim 161.2$ cm)	159.2	65.3	49.1	83.8	38.8	30.4	25.8	1436
3 ( $\sim 165.5$ cm)	163.3	67.6	46.2	84.1	35.9	30.7	25.4	1495
4 ( $> 165.5$ cm)	169.3	71.1	42.6	83.7	35.0	31.4	24.8	1585

<sup>a</sup> Least-squares means estimated through linear regression models which included age as an independent variable (except in model estimating mean age).

<sup>b</sup> Based on 24-h dietary recall questionnaire.

Table 2 Age-adjusted relative risk<sup>a</sup> of cancer according to quartile of stature by sex, United States, 1971-1984

Cancer site	No. of cases	Stature quartile <sup>b</sup>			
		1	2	3	4
Males					
All sites					
Age adjusted	460	1.0	1.5 (1.2-1.9) <sup>c</sup>	1.4 (1.1-1.8)	1.4 (1.1-1.9)
Multivariate <sup>d</sup>	341	1.0	1.6 (1.2-2.3)	1.5 (1.2-2.3)	1.6 (1.1-2.3)
Lung	114	1.0	1.4 (0.8-2.3)	1.6 (0.9-2.3)	1.1 (0.6-2.0)
Prostate	95	1.0	1.2 (0.7-2.1)	1.1 (0.6-1.9)	1.1 (0.6-2.0)
Colorectum	62	1.0	1.8 (0.9-3.6)	1.8 (0.9-3.8)	2.1 (1.0-4.5)
Bladder	27	1.0	0.8 (0.3-2.4)	2.0 (0.8-5.1)	0.7 (0.2-2.8)
Other sites (combined)	170	1.0	1.7 (1.1-2.5)	1.3 (0.8-2.0)	1.7 (1.1-2.7)
Females					
All sites					
Age adjusted	399	1.0	1.0 (0.8-1.3)	1.1 (0.8-1.4)	1.2 (0.9-1.5)
Multivariate	302	1.0	1.2 (0.9-1.6)	1.2 (0.9-1.7)	1.1 (0.8-1.6)
Breast	122	1.0	1.4 (0.9-2.4)	1.3 (0.8-2.3)	2.1 (1.2-3.4)
Colorectum	67	1.0	0.8 (0.4-1.6)	1.0 (0.5-1.9)	1.6 (0.8-3.0)
Uterus (corpus)	30	1.0	1.5 (0.5-4.4)	3.7 (1.4-9.3)	1.0 (0.2-3.9)
Cervix	20	1.0	0.5 (0.1-1.8)	1.2 (0.4-3.5)	0.5 (0.1-2.2)
Other sites (combined)	168	1.0	1.0 (0.7-1.4)	0.8 (0.5-1.2)	0.7 (0.4-1.2)

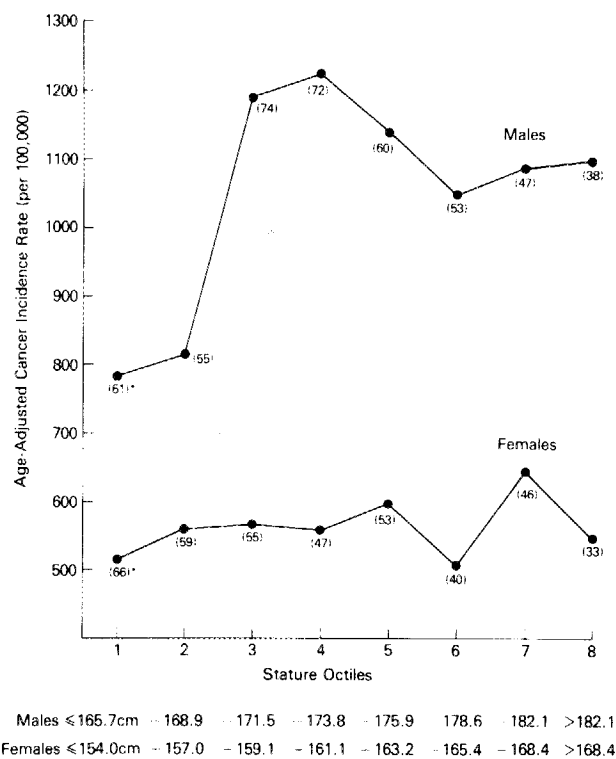
<sup>a</sup> Relative risk was estimated through proportional hazards regression modeling which included age as a continuous independent variable.<sup>b</sup> Quartile boundaries same as for Table 1.<sup>c</sup> Numbers in parentheses, 95% confidence limits.<sup>d</sup> Proportional hazards regression model included age, race, cigarette-smoking status, family income, and BMI for both sexes as well as menopausal status, age at menarche and menopause, and parity for women. Only persons with complete data for all variables were included in the analysis.

Fig. 1. Age-adjusted cancer incidence rate (/100,000 person-years) according to octile of stature by sex, United States, 1971-1984.

somewhat elevated compared to controls (27). One report of a positive relationship between stature and lung cancer (especially in men) (15) was followed by a supportive analysis (16) and two other reports showing no association (29, 30). Our demonstration of a positive stature-breast cancer relationship corroborates most prior studies of that site, including one which demonstrated a strong correlation internationally between age-specific stature of children and adolescents and breast cancer rates (33), while the approximately 2-fold increase in colorectal cancer risk among the tallest participants has not been previously

observed. There have also been reports of increased stature among patients with acute lymphocytic leukemia (18), osteogenic sarcoma (19), or Hodgkin's disease (17), but these sites could not readily be evaluated in the present study of adults.

Several plausible explanations for the observed stature-cancer relationship can be suggested based upon the biology of human growth. Adult stature is determined primarily by two factors: heredity (e.g., parental stature) and nutritional exposures (e.g., energy and other macronutrient intake) experienced during development. It is possible that some hereditary factor resulting in short stature may lead to decreased cancer risk. For example, the relevant gene(s) or gene product(s) might inhibit activation of a cellular protooncogene or otherwise diminish the risk of malignant transformation at the cellular level. Alternatively, short individuals may be exposed to carcinogens to a lesser extent than taller persons because of dissimilar behaviors or environments. Of the factors available for assessment in this study (e.g., cigarette smoking), most differed only slightly between stature groups and could not readily explain the observed effects for all the cancer sites involved. Only energy intake, a nutritional factor associated with carcinogenesis in rodents, increased linearly and dramatically with stature in both sexes. However, the risk of developing cancer did not increase with adult caloric intake [possibly because of the influence (not controlled for) of other factors involved in energy metabolism such as body mass, physical activity, and basal metabolic rate], and the relative risk of all-sites cancer associated with stature was unchanged after adjustment for adult kilocalorie or total fat intake. Therefore, while average energy intake in adulthood increased with stature, it did not confound the relationship between stature and cancer.

An alternative hypothesis involves the effects of early nutrition on growth and carcinogenesis. Calorie and macronutrient intake during development greatly affect growth and stature in humans (34, 35). Early caloric restriction is also known to reduce tumor incidence in rodents, although later restriction is also protective (10). In well-nourished human populations such as this cohort, it may only be in the shortest categories that there are individuals who have experienced calorie or macronutrient restriction during growth sufficient to affect both their

Table 3 Age-adjusted relative risk<sup>a</sup> of cancer according to quartile of leg length and sitting height in males, United States, 1971-1984

	No. of cases		Quartile <sup>b</sup>			
			1	2	3	4
All sites						
Age adjusted	460	LL <sup>c</sup>	1.0	1.4 (1.1-1.8) <sup>d</sup>	1.4 (1.1-1.9)	1.6 (1.2-2.1)
		SH	1.0	1.0 (0.8-1.3)	1.0 (0.8-1.3)	1.1 (0.8-1.4)
Multivariate <sup>e</sup>	341	LL	1.0	1.4 (1.0-1.8)	1.3 (1.0-1.8)	1.5 (1.1-2.0)
		SH	1.0	1.1 (0.8-1.5)	1.1 (0.8-1.5)	1.2 (0.9-1.7)
Lung	114	LL	1.0	1.3 (0.8-2.2)	1.2 (0.7-2.1)	1.6 (0.9-2.7)
		SH	1.0	1.0 (0.6-1.6)	1.0 (0.6-1.6)	0.6 (0.3-1.1)
Prostate	95	LL	1.0	1.1 (0.7-1.9)	0.9 (0.5-1.6)	1.2 (0.7-2.2)
		SH	1.0	1.1 (0.6-1.8)	1.1 (0.6-1.9)	1.2 (0.6-2.3)
Colorectum	62	LL	1.0	1.1 (0.5-2.2)	1.5 (0.8-3.0)	1.5 (0.7-3.0)
		SH	1.0	1.1 (0.6-2.0)	1.4 (0.7-2.8)	1.4 (0.6-3.0)
Bladder	27	LL	1.0	1.4 (0.5-3.9)	0.9 (0.3-2.8)	1.1 (0.4-3.6)
		SH	1.0	0.8 (0.2-2.3)	2.1 (0.8-5.5)	1.2 (0.3-4.0)
Other sites (combined)	170	LL	1.0	1.8 (1.1-2.9)	2.0 (1.3-3.2)	2.0 (1.2-3.2)
		SH	1.0	1.0 (0.7-1.5)	0.9 (0.5-1.3)	1.2 (0.7-1.8)

<sup>a</sup> Relative risk was estimated through proportional hazards regression modeling which included age as a continuous independent variable.<sup>b</sup> Quartile boundaries are 79.9, 82.9, and 86.1 cm for leg length, and 88.2, 91.0, and 93.4 cm for sitting height.<sup>c</sup> LL, leg length; SH, sitting height.<sup>d</sup> Numbers in parentheses, 95% confidence limits.<sup>e</sup> Proportional hazards regression model included age, race, cigarette-smoking status, family income, and BMI. Only persons with complete data for all variables were included in the analysis.Table 4 Age-adjusted relative risk<sup>a</sup> of cancer according to quartile of leg length and sitting height in females, United States, 1971-1984

	No. of cases		Quartile <sup>b</sup>			
			1	2	3	4
All sites						
Age adjusted	399	LL <sup>c</sup>	1.0	1.0 (0.8-1.3) <sup>d</sup>	0.9 (0.7-1.2)	1.2 (0.9-1.6)
		SH	1.0	0.9 (0.7-1.1)	1.2 (0.9-1.6)	1.1 (0.8-1.4)
Multivariate <sup>e</sup>	302	LL	1.0	1.0 (0.8-1.4)	0.9 (0.7-1.2)	1.2 (0.8-1.6)
		SH	1.0	0.8 (0.6-1.1)	1.2 (0.9-1.5)	1.0 (0.7-1.4)
Breast	122	LL	1.0	1.1 (0.6-1.8)	0.9 (0.5-1.6)	1.6 (1.0-2.6)
		SH	1.0	0.9 (0.5-1.4)	1.2 (0.8-2.0)	1.3 (0.7-2.2)
Colorectum	67	LL	1.0	1.1 (0.5-2.1)	1.1 (0.6-2.2)	1.4 (0.7-2.8)
		SH	1.0	0.7 (0.4-1.4)	1.1 (0.5-2.1)	1.4 (0.7-3.0)
Uterus (corpus)	30	LL	1.0	0.3 (0.1-1.3)	1.2 (0.5-2.8)	1.1 (0.4-2.8)
		SH	1.0	0.9 (0.3-2.4)	1.4 (0.5-3.5)	0.9 (0.3-3.2)
Cervix	20	LL	1.0	2.6 (0.5-13.4)	3.0 (0.6-14.8)	3.8 (0.8-18.3)
		SH	1.0	0.4 (0.1-1.5)	0.3 (0.1-1.4)	1.0 (0.3-3.1)
Other sites (combined)	168	LL	1.0	1.1 (0.7-1.6)	0.7 (0.5-1.1)	0.8 (0.5-1.2)
		SH	1.0	1.0 (0.6-1.4)	1.4 (0.9-2.0)	0.8 (0.5-1.4)

<sup>a</sup> Relative risk was estimated through proportional hazards regression modeling which included age as a continuous independent variable.<sup>b</sup> Quartile boundaries are 73.2, 75.9, and 78.9 cm for leg length, and 82.8, 85.3, and 87.6 cm for sitting height.<sup>c</sup> LL, leg length; SH, sitting height.<sup>d</sup> Numbers in parentheses, 95% confidence limits.<sup>e</sup> Proportional hazards regression model included age, race, cigarette-smoking status, family income, BMI, menopausal status, age at menarche and menopause, and parity. Only persons with complete data for all variables were included in the analysis.

ultimate stature and future risk of cancer. Our observation of a threshold effect for stature is consistent with this premise. Early restriction may operate through endocrine or other physiological mechanisms (36), or by diminishing cell proliferation (*i.e.*, mitotic activity) during development, thereby reducing cell number and organ size, or conceivably, the risk of an initiating event (37, 38).

Several of the present findings support an early nutrition hypothesis. The greater effect observed in men is consistent with evidence suggesting increased sensitivity of stature in this sex to dietary restriction, compared to women (39, 40). Also, we demonstrated a stronger association for the leg length component of stature than for sitting height. Leg length is more sensitive to environmental influences, especially those occurring

during adolescence, than is sitting height (41). Finally, we demonstrated a greater association with stature for breast cancer than for colorectal cancer in women, a finding which parallels previous observations of a slower upward drift of breast cancer incidence rates (compared to colorectal cancer rates) among women who migrated from Japan to the United States (42, 43). These studies have been interpreted as implicating early life factors, such as early nutrition, to a greater degree in the pathogenesis of breast cancer than in colon cancer, for which exposures occurring later in life (including adult diet) may play a relatively more important role.

In summary, although these findings will require confirmation from other investigations (including some which have previously evaluated the relationship between cancer and rela-

tive body weight indices), they support a role for early nutritional exposure in subsequent cancer risk. Studies with information concerning both childhood growth and nutrition and subsequent cancer would be particularly informative and should be examined. The potential for providing insight into the mechanisms of carcinogenesis warrants further investigation of this area.

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